

Easy Pathology

— Dr. Abdelrahman Khalifa —

Chapter 1

Cell Injury

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Cell Response to injury

Morphological & functional changes of cells following stressful conditions

- Adaptations
- Reversible injury (degenerations)
- Irreversible injury (necrosis & apoptosis)
- Accumulations & tissue deposits

Adaptation

1. Atrophy

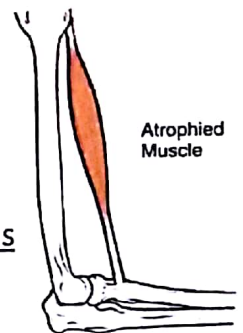
Decreased size of organ due to decreased size and/or number of cells

• Physiological

- Atrophy of thymus after puberty
- Lymphoid tissue, & appendix in old age
- Post menopausal atrophy of ovaries → ↓ estrogen.

• Pathological

- Ischemic atrophy (chronic ischemia) → tissue atrophy
- Pressure atrophy e.g. Aortic aneurism → atrophy of vertebral bodies
- Neurogenic (Poliomyelitis, paralysis)
- Disuse atrophy (fractured limb)
- Hormonal (e.g. Pituitary diseases with lack of TSH → thyroid atrophy)
- Nutritional protein deficiency (e.g. marasmus) *food with out Proteins*



Morphology: Decreased cell size – decreased organelles (mitochondria, ER, myofilaments) – autophagy vacuoles (e.g. lipofuscin)



Aplasia : Failure of organ development which remain small in size

Agenesis: Failure of organ formation (complete absence)

2. Hypertrophy

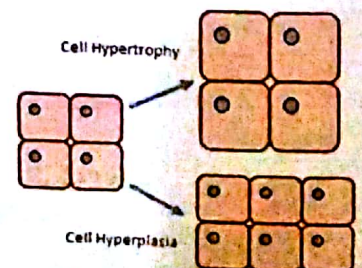
Increased size of organ due to increased size of cells (in permanent cells)

• Physiological

- Exercise → muscle hypertrophy
- Pregnancy → uterine hypertrophy

• Pathological

- Hypertension, valve diseases → cardiac hypertrophy
- Pyloric stenosis → gastric hypertrophy
- Compensatory in one kidney after nephrectomy of the other one.
- Urethral obstruction → bladder hypertrophy



Morphology: Increased cell size – Increased nuclear size

Kidney not forming cell and not muscular cell

3. Hyperplasia

Increased size of organ due to increased number of cells

Site: Labile & stable cells (cells with mitotic ability), NOT in permanent cells

• Physiological

- Hormonal : Breast (during lactation) , Uterine muscle (pregnancy)
- Growth factors (Compensatory) : e.g. Liver (partial hepatectomy) , Kidney (nephrectomy)

• Pathological

○ Hormonal :

- Mammary hyperplasia (fibrocystic disease)
- Endometrial hyperplasia → ↑ estrogen → precancerous
- Prostatic hyperplasia
- Thyroid hyperplasia in Goiter (elevated TSH)
- Bone marrow , following blood loss (erythropoietin)

○ Reactive : L.N. hyperplasia following infection

○ Irritation : urothelial hyperplasia in bilharziasis (Brunn's nest)

Hyperplasia may occur with hypertrophy in some cases



It is reversible

It is precancerous in some pathological cases (endometrial, mammary)

	Hyperplasia	Neoplasia (Tumor)
Aetiology	Initiated by a cause (stimulus)	Multi-factorial
Course	Self-limited	Uncontrolled
Fate	-Reversible (after removal of the cause) -Could be precancerous (endometrial)	Irreversible

4. Metaplasia

Transformation of a cell into another type of cell

mechanism: Abnormal stimuli (Irritant) → Reprogramming of stem cells → new type

• Epithelial

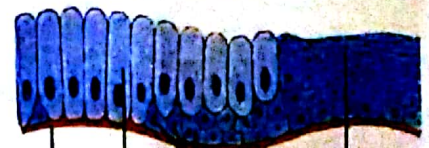
○ Squamous metaplasia

- Smoking → Squamous metaplasia of Bronchi
- Shistozoma → Squamous metaplasia of Bladder
- Stones → Squamous metaplasia of urinary or gall Bladder
- Chronic nasal polyp
- Chronic Cervicitis

○ Columnar metaplasia

St. Sq. ▪ Esophagus: Barret's esophagitis in case of GERD

simplex ▪ Stomach: Intestinal metaplasia in Chronic gastritis



Barrett's esophagus

Bladder: Cystitis cystic → cystitis glandularis → Cause *Balantidia*.

• Mesenchymal *not epithelium another thing*

○ Osseous → *per bone*

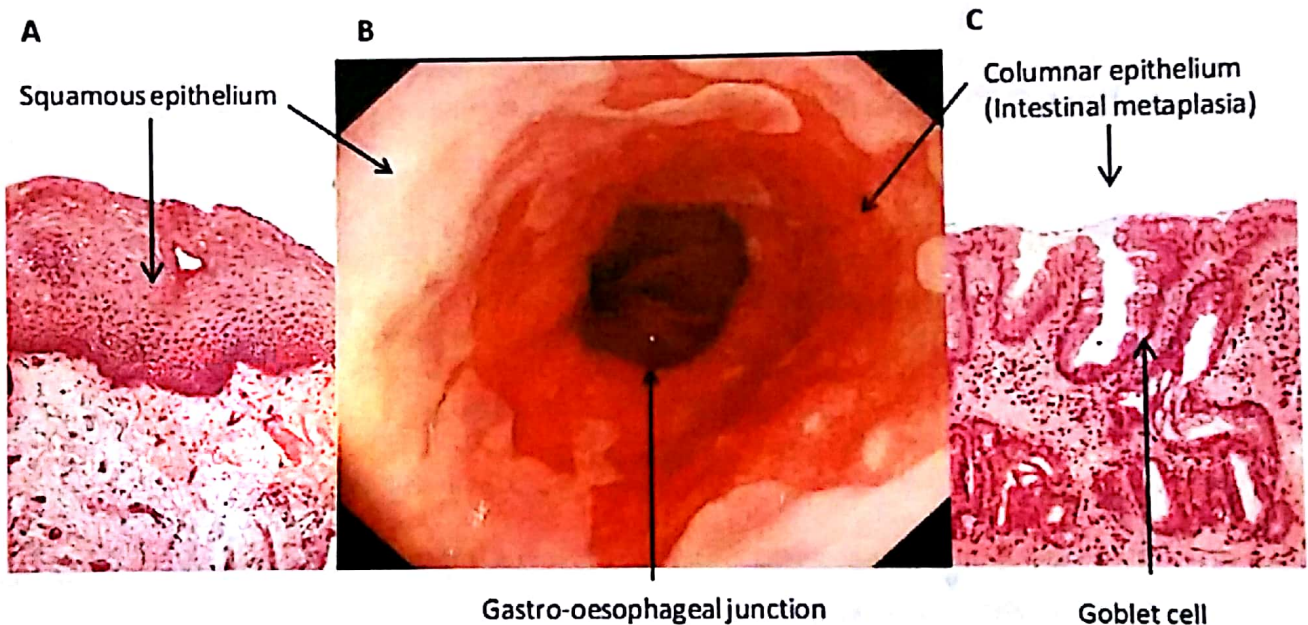
- Myositis ossification in muscle
- Ossification of calcified areas
- Ossification of tumor stroma

○ Cartilagenous metaplasia of Splenic capsule (in splenomegaly) → *Balantidia* *Chloroform* *Ven*

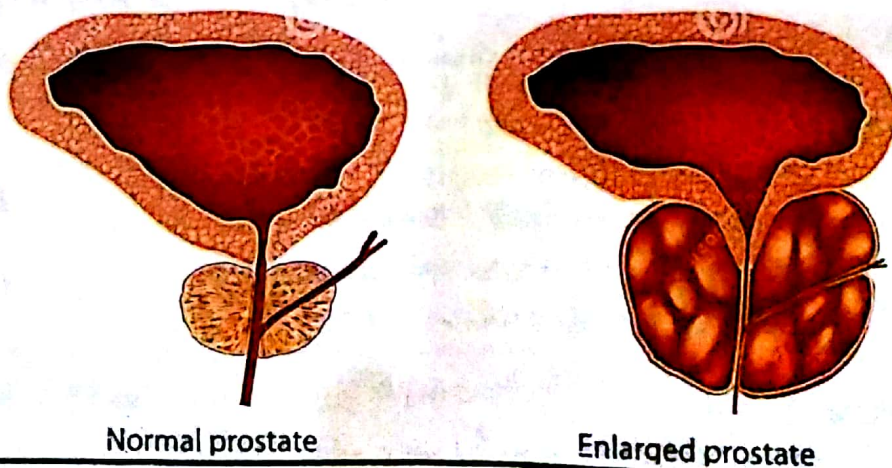
○ Myeloid metaplasia in case of blood loss (extramedullary hematopoiesis)

Same bone marrow in other site than bone.

N.B. Adaptations are reversible



Benign Prostatic Hyperplasia



Reversible injury

Causes:

- Hypoxia (decrease oxygen supply)
 - ↓ BLS → Ischemia , heart failure
 - ↓ O₂ → Anemia, or heart diseases
- Infections : viral – bacterial – fungal – parasitic ...etc. { *micro organisms* }
 - micro organisms* → *viruses* *D. a. q. m.* *Parasite*
- Immunological: – Ag-Ab reaction, Autoimmune → *hyper sensitivity*
- Physical: e.g. Temperature- trauma – Radiation
- Chemicals: Acids – Alkalis – Poisons – drugs –
- Genetic disorders *alk(1) ...*
- Nutritional : protein deficiency (*new cells*) (marasmus, Kwashiorkor) – excess fat

Factors affecting response to injury:

Type of injury (severity & duration) - Type of cells (labile – stable – permanent)
Skeletal muscle resists hypoxia for 2-3 hours, Cardiac muscle resists for 2-3 minutes

Pathogenesis



- ↓ ATP → Defective cell membrane transport (Na-K pumps) →
↑ intracellular Sodium (Na) → water influx → Cell Swelling & organelles swelling
mitochondria damage (appear as amorphous deposits or vacuoles) :
- Release of free radicals (Active oxygen metabolites) → damage of proteins, lipid, & DNA
 - Release of Cytochrome C → apoptosis
 - ATP depletion



↓ ATP → Calcium influx → ↑ intracellular Ca → activate damaging enzymes:

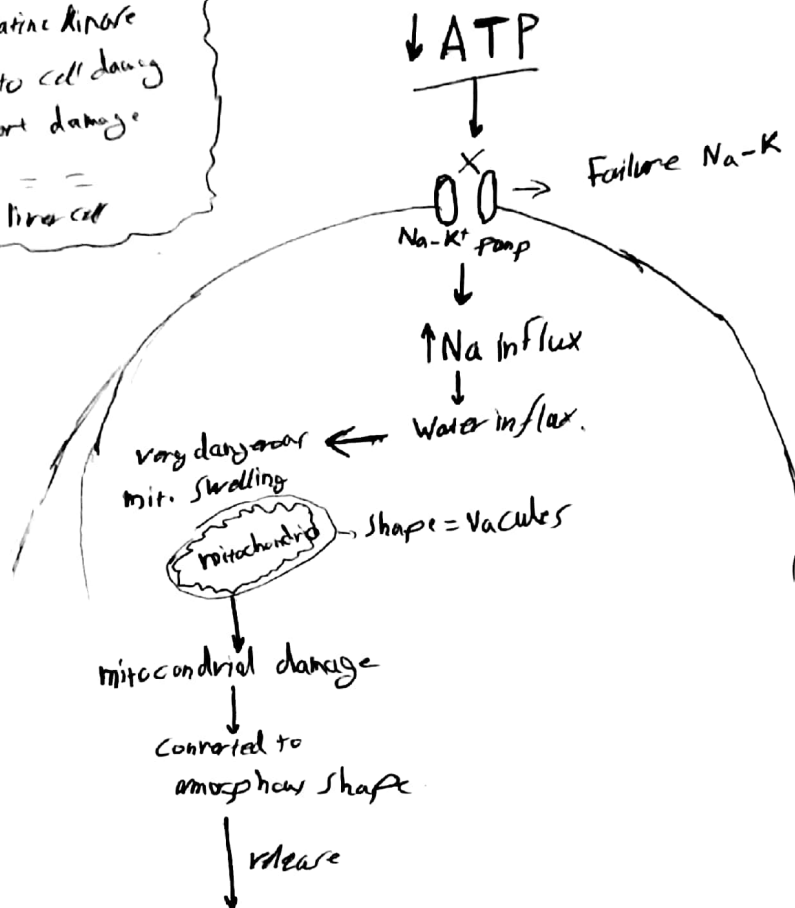
- ATPase → more ATP loss
- Protease → damaged proteins & nucleoprotein
- Caspase → Cell apoptosis
- Phospholipase →
 - membranes damage: → Myelin figures
 - Phagocytosed
 - Bind with Ca → saponification
 - Release of intracellular enzymes to blood
 - Mitochondrial & lysosomal damage
- Endonuclease → damage of nucleus, DNA, RNA
 - *Pyknosis* (condensation)
 - *Karyorrhexis* (fragmented)
 - *Karyolysis* (dissolution)



↓ ATP → Anaerobic glycolysis → *lactic acidosis* → activates enzymatic digestion

- Pathogenesis - (mechanism of cell injury)

↑ Creatine Kinase
due to cell damage
in heart damage
↑ AST = =
↑ ALT liver cell



① - Reactive oxygen species = Free Radical → attached to other organism and destroyed it

② - Cytochrome C → Caspases → Apoptosis kill cells

more decrease in ATP → loss of ATP → ↑ Ca Intra cellular → ④ damaging enzymes → destroyed enzymes → fragment cell



Points of no return = irreversible state

membrane damage
mitochondrial damage
nuclear damage

For Page 4

① destroyed C.m

- Myelin figures

contact with Ca²⁺

so denaturation ⇒ destroyed C.m

so will release all these

enzyme and destroyed

another cells

③ lysosomal enzyme activated

by ↑ Lactic acid ⇒ that ↑ pH

We anaerobic system to produce

energy → so more activation of lysosomes

① - Protease → cytoplasmic and nuclear

② - caspases → Apoptosis

③ - phospholipase → cell membrane

④ - endonuclease → damage DNA RNA

⑤ - endonuclease

⑥ - endonuclease

⑦ - endonuclease

⑧ - endonuclease

⑨ - endonuclease

⑩ - endonuclease

⑪ - endonuclease

⑫ - endonuclease

⑬ - endonuclease

⑭ - endonuclease

- ① **Points of no return:** The critical steps that lead to irreversible changes are permanent Mitochondrial dysfunction & cell membrane damage.

Hydropic degeneration (cloudy swelling)

Definition: Reversible cell injury, with intracellular water vacuoles

Causes: causes of cell injury (enumerate) but mild for short duration

Pathogenesis:

Hypoxia → Loss of ATP → Failure of sodium pumps → Sodium & water influx → Cell swelling

Site: highly specialized organs (e.g. Liver, & kidney) *mitochondrial swelling without damage*

Gross:

- ① - Size : swollen
- Surface : smooth
- Cut section : bulging
- Color : pale bloodless (compressed capillaries)
- Consistency : Soft or ~~firm~~ *firm*

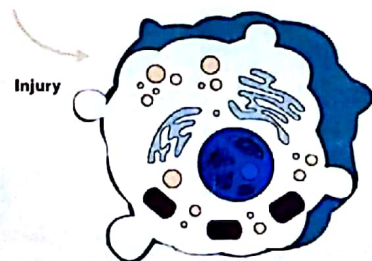


Microscopic:

- ① - Cells: Swollen – intact membrane – cytoplasmic vacuoles (swollen ER)
- Stroma: compressed capillaries

E/M (ultrastructure):

- Cell swelling
- Blebs at the surface
- Loss of microvilli in some epithelial cells
- Ribosomes are detached
- Mitochondria & ER are swollen
- Chromatin clumping
- Loss of cellular attachment



© Stud

Fate:

If the cause is removed → cells return to normal
If not → necrosis

Clinical:

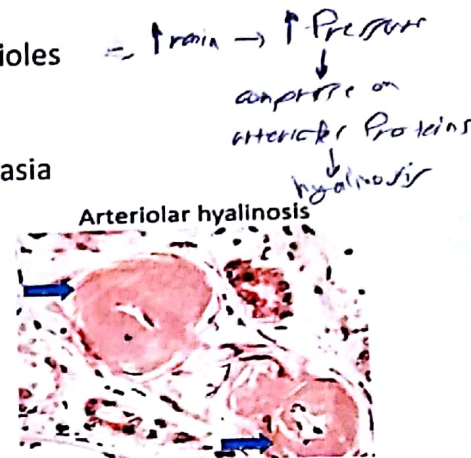
Liver → hepatomegaly and jaundice due to occlusion of bile canaliculi
Intestine → diarrhea

Hyalinosis

Microscopic change in tissue proteins, which became glassy, homogenous & eosinophilic.

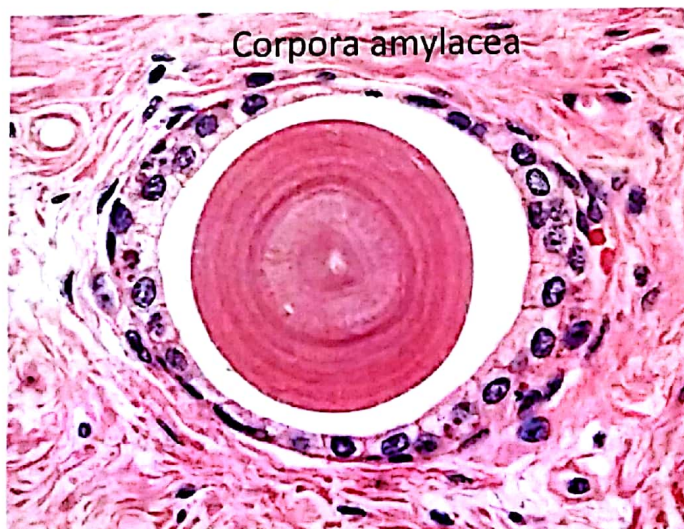
1. Extracellular hyalinosis (CAPS)

1. **Chronic Glomerulonephritis** : hyalinosis of arterioles
2. **Arteriosclerosis** : in malignant hypertension
3. **Prostate**: **Corpora amylacea** in prostatic hyperplasia
4. **Scar** : hyalinosis of collagen in old scar



2. Intracellular hyalinosis: (4 P)

- **Plasma cell** → accumulated Ig → **Russell body**
- **Parenchyma of liver** (in alcoholism) → **Mallory body**
- **Proximal convoluted tubules** (in proteinuria) → **Hyaline bodies**
- **Pneumonia, or Diphtheria** : → Toxemia → **Abdominal muscle injury** → Lactic acidosis → hyaline change of muscle proteins → **homogenous pink & loss of striations** → grossly became **pale, friable, and weak** (**Zenker's degeneration**)



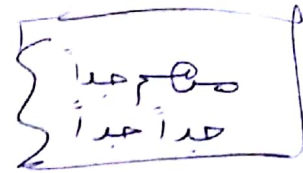
Mucoid degeneration

myxoid degeneration

Mucoid changes	Myxoid changes
Intracellular Accumulation of mucinous material (glycoprotein) in epithelial cells	Extracellular Accumulation of mucinous material (glycoprotein) in connective tissue
Catarrhal inflammation Mucoid Carcinoma Muco-Cele	Myxoid changes in tumors - Myxoedema

Irreversible injury

NECROSIS



Definition: Morphological changes following death of group of cells inside living tissue, directly, or following degeneration.

Pathological picture:

Gross: Yellowish, swollen, & pale → then soft shrunken

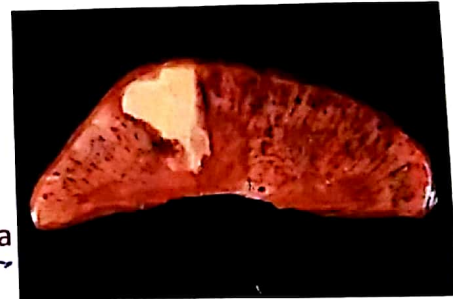
Microscopic:

- Outlines: early preserved, but no cytoplasmic details
- Cytoplasm: eosinophilia (due to loss of RNA & denaturated proteins)
- Nucleus: Pyknosis – Karyorhexis – Karyolysis

Morphological Types:

Coagulative necrosis:

- The most common type of necrosis
- Tissue resembles "Boiled meat"
- Cause: Hypoxia is the most common, mainly ischemia
- Gross: dead area is "firm", and "pale"
- Microscopic: Architecture is preserved in early stage (Cell boundaries and stroma, but nuclei are lost)
- e.g.: Myocardial infarction (MI), Renal infarction



Caseation necrosis:

- Tissue resembles Casine "Cheese"
- occur in Tuberculosis (T.B.)
- Cause: Hypersensitivity against bacteria → tissue caseation
- Gross: dead area is "soft cheesy, granular", and "yellowish white"
- Microscopic: tissue became structureless homogenous, with granular areas + granuloma of TB



Liquifactive necrosis:

- Cause: 1- Proteolytic enzymes in Abscess
2- Brain infarction
3- Liquifactive enzymes of amoeba
- Gross: dead area is "liquified", and "yellowish"

- **Microscopic:** tissue became structureless homogenous (complete liquefaction) → cystic change or cavitation
Surrounded by inflammatory cells & macrophage
Surrounded by fibroblasts or glial cells (in CNS)

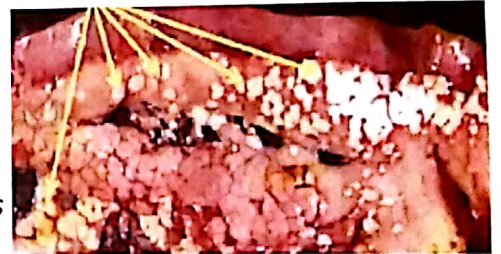
Fat necrosis:

- It is necrosis of "Fat cells" in adipose tissue
- **Cause: 1- Traumatic**
- Trauma of subcutaneous fat, may lead to ruptured fat cells, and deposition of calcium salts in the area of fat necrosis, giving hard palpable mass.

2-Enzymatic

Acute pancreatitis → release of lipase → fat cell necrosis in omentum
→ release of fatty acids (FA) → FA + intracellular Ca → Calcium salt (Saponification)

- **Gross:** dead area is "Chalky", and "white"
- **Microscopic:** - Injured fat cells (shadows)
- Calcification (basophilic)
- inflammatory cells and giant cells
- e.g. : - Traumatic fat necrosis of the breast
- Enzymatic fat necrosis in case of acute pancreatitis



in case of hard mass in the breast of young female, traumatic fat necrosis should be taken in consideration before diagnosis of cancer breast.

Fibrinoid necrosis:

- Autoimmune → Deposition of fibrin-like material
- e.g. Vasculitis, malignant hypertension, Aschoff nodule
- homogenous eosinophilic deposit in the wall



N.B. Autolysis: damage of cell by its own lysosomal enzymes mainly postmortem

Fate of necrosis

- Inflammation: All types of necrosis initiates inflammatory reaction
- Regeneration
- Fibrosis: depends on type of tissue and severity of injury
- Capsulation: fibrosis around the periphery of necrotic area — due to presence of vital cells
- Calcification: deposition of calcium salts inside dead tissue
- Putrefaction (Gangrene) : if infected with certain types of bacteria

Apoptosis

Definition: Morphological change following death of single cell or group of cells, by energy-dependant programmed pathway.

Causes:

- Physiological (mainly Hormonal deprivation)
 - o Menstruation , hormonal-induced apoptosis of endometrium
 - o Embryogenesis, absence of some embryonic structures
 - o After weaning, regression of mammary gland
 - o Thymus atrophy
- Pathological (disease-induced)
 - o HCV → Liver cell apoptosis (councilman body)
 - o HLA mismatch → graft rejection
 - o Chemotherapy → tumor cell apoptosis
 - o Cancer prostate (after orchiectomy) →
 - o Cerebral apoptosis (in Alzheimer)



Mechanism:

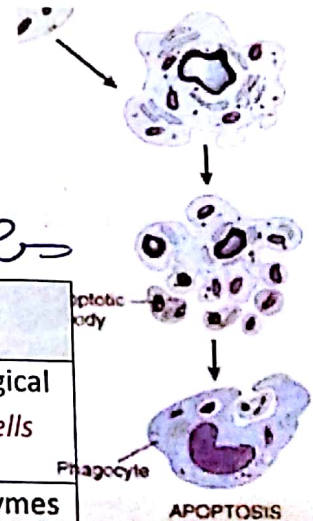
Extrinsic:

Expression of FAS receptors on cell → bind with FAS-L on lymphocytes → activation of caspases (caspase 8) → apoptosis

Intrinsic: mediated by regulation of Bcl2 family genes → activation of Bax & bak, inhibition of Bcl → increased mitochondrial permeability → release of cytochrome C → activation of caspases

Morphological changes :

- Cell shrink in size & convoluted of membrane
- Condensation of chromatin or fragmented
- Condensed organelles
- Cell divides into smaller apoptotic bodies
- Apoptotic bodies are phagocytosed



	<u>Necrosis</u>	<u>Apoptosis</u>
causes	Pathological injury <i>group of cells</i>	Physiological or pathological <i>Single cell or group of cells</i>
mechanism	Controlled by ATP depletion	Controlled by genes, enzymes and hormones
microscopic	-large cell -damaged membrane - damaged organelles -damaged nucleues	-Small cell -intact membrane with blebs -condensed organelles -condensed chromatin
inflammation	present	absent
fate	(enumerate)	phagocytosis

DNA irreversible damage
+ ↓ activation

P₅₃ → ⊖ BCL

+ Bax
+ Bak

↑ mitochondrial permeability
for cytochrome c
by formation of



+ Caspase 9

⊕ Apoptosis

+ T-lymphocyte (TF)

+ Fas (L)

+ Fas (R)

+ Caspase 8 → ⊕ apoptosis

For Page
9

Gangrene

Senile Massive tissue necrosis, followed by putrefaction

Putrefaction: Breakdown of dead tissue by saprophytic bacteria → release of H₂S (offensive gas) → formation of Iron Sulphide (black color)

Dry gangrene

Gangrene Caused by arterial occlusion (e.g. Thrombus) → followed by tissue dryness (mummification) → then putrefaction e.g. **Senile dry gangrene of the foot**

Pathogenesis:

- Ischemia (P.F. : Atherosclerosis, & H.F.)
- Tissue necrosis & inflammatory edema
- Dryness (mummification)
 - Edema is **drained** by lymphatics, and veins.
 - **Evaporation** of tissue fluid. No more fluid supply (ischemia)
- Putrefaction & spread
- **Line of demarcation**
 - Putrefaction stop at the area with **good blood** supply
 - The **red line** separating healthy tissue from putrefied tissue
- **Granulation tissue**
 - Healing starts from line of demarcation → extend distally
- **Line of separation**
 - The line separating GT from remaining unhealed putrefied tissue
- **Self separation (amputation)**
 - The remaining dead tissue begin to separate spontaneously leaving **conical stump**



Dry gangrene



moist gangrene

	Dry gangrene	Moist gangrene
Aetiology	Arterial occlusion Atherosclerosis – Burger -Reynaud	Arterial occlusion in moist areas Moist tissue (mouth, cervix) Intestine (strabgulation) D.M. (excess inflammation & fluid) Bed sores (arterial & venous compression)
Ischemia	prsent	present
Edema	present	more
Mummification	present	No mummification
Putrefaction and spread	Slow	Rapid
Line of demarcation	present	No line of demarcation
Line of separation	Present	No line of separation
Self separation	present	No self separation
Complications	Mild Toxemia (sapremia)	Rapid spread Severe Toxemia (sapremia)

Gas gangrene

Putrefaction of muscles, through infection of deep wounds

Aetiology:

1. Infected deep wounds by anerobic Cholestidia from soil
2. Infected wound during colon surgery

Pathogenesis :

3. Muscle necrosis + Hemolysis → Red dead muscle
4. Putrefaction → extensive gas (H₂S) accumulation + Black swollen muscle
5. Excessive gas → Crepitation

Fate : Severe toxemia



Fatty changes

Definition: Reversible cell injury, showing neutral fat (triglycerides) accumulation

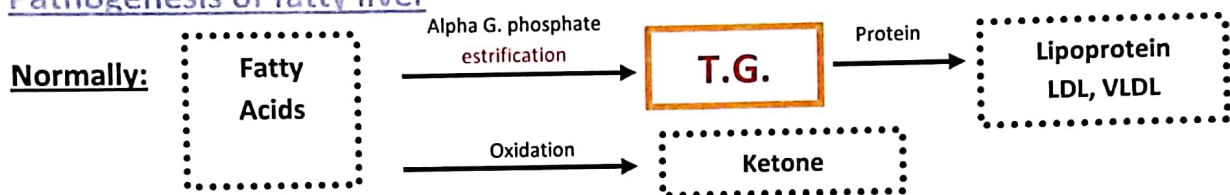
Causes: causes of cell injury, Mostly: hypoxia, bacterial toxins, chemicals

Sites: Liver (most common) – Heart - Kidney

causes of fatty liver

- Hyperlipidemia (Congenital – Obesity – DM – Alcohol the most common)
- Drugs & toxins (CCL4, Steroides) *or diseases* *Carbon Tetrachloride*
- Diseases (H.F. – Anemia – TB)

Pathogenesis of fatty liver



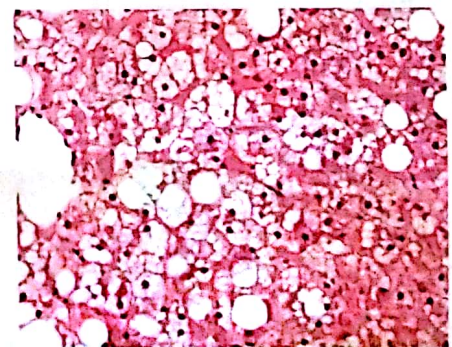
In Fatty changes:

- Increased F.A. intake, or synthesis → T.G.
- Increased F.A. estrification → T.G.
- Decreased F.A. oxidation → increased F.A. → T.G.
- Decreased protein carriers → T.G. accumulation
- Decreased Lipoprotein excretion



Gross:

- Size : enlarged with tense capsule
- Surface : smooth with rounded borders
- Cut section : bulging and greasy
- Color : Yellow
- Consistency : Soft Greasy



Microscopic:

- **Cells:** swollen, intact membrane, with clear cytoplasmic vacuoles (microvesicular steatosis) → large single vacuole (macrovesicular) → signet ring cells
- **Ruptured fat cells** → Fat cyst & lipogranuloma
- **Staining:** Sudan III, IV, black, Osmic acid, Oil red

orange

Black

stained

Fatty changes of the Heart

Diffuse fatty change	Patchy (focal) fatty change
Severe	Mild
In cases of Toxemia , severe hypoxia	In case of Anemia
Yellow in color	Yellow & brown (Tabby cat/ tiger skin)

Fatty infiltration

Accumulation of fat inside connective tissue due to obesity or chronic diseases.
(e.g. Heart , pancreas)

Cholesterol deposition:

- Macrophages (in old hemorrhage or necrosis) → foam cells
- Atherosclerosis (in macrophages 'foam cells' inside arterial wall)
- Xanthoma (in macrophage of Skin in cases of hyperlipidemia)



Cholesterosis: accumulation of cholesterol in G.B. epithelium

Melanin pigment

Tyrosine → converted into brown melanin (by tyrosinase enzyme) → engulfed by macrophage (melanophores)

Hyper pigmentation

- Generalized
 - **Chloasma**: butterfly color in face and breast (estrogen-progesteron)
 - **Adisson's**: due to increased ACTH, MSH
- Localized Patchy
 - **Sun Freckle** - *كحل*
 - **Melanocyte tumors** (nevus- melanoma) *جذام*



Hypo pigmentation

- Generalized **Albinism**: congenital 'autosomal-recessive' tyrosinase deficiency
- Localized Patchy
 - **Leukoderma** which is congenital or secondary to Syphilis or Leprosy
 - **Vetiligo** which is an autoimmune dis *زحري* *الجذام*

Glycogen

In D.M.: due to abnormal glucose metabolism

Glycogen storage disease: genetic, metabolic disease. Accumulation of glycogen in Liver, muscle, and heart. Cells show clear cytoplasm

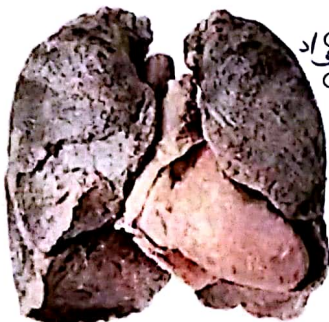
Stain → PAS + Best's carmine

Hemosiderin *deposited brown*

F.A.S

Ferretin accumulates inside tissue macrophage (yellowish brown) → stains blue with Perl or Prussian blue

Localized Hemosiderosis — <i>H₂O₂ macrophages</i>	Generalized hemosiderosis —
• Skin hemorrhage (bruise) green <u>billiverdin</u> → brown <u>hemosedrin</u> • Lung congestion → macrophage engulf hemosedrin → <u>H.F.</u> cells (brown induration of lung)	• Hemolysis • Repeated transfusion • Increased Iron intake Engulfed by macrophage of <u>Liver</u>, <u>spleen</u>, <u>B.M.</u>, & <u>L.N.</u> ↓
1ry Hemochromatosis — <i>كروماتوزيس</i> (bronzed D.M.)	2ry hemochromatosis
Increased iron absorption (autosomal dominant) → deposition in <i>تجمع الحديد في الكبد والبنكرياس</i> • — Pancreas → DM <i>مرض السكري</i> • — Liver → pigmented cirrhosis • — Skin → bronzed color	Prolonged generalized hemosidrosis — <i>hemolysis</i> — Liver — = — spleen — = — DM — = — LN

Exogenous Pigments*in long Term Tissue deposit*

1/2 **Tattoo** (Indian ink → engulfed by macrophage)

○ **Anthracosis** (Black Carbon deposition e.g. smoking, pollution → engulfed by lung macrophages → black color in lung and hilar L.N.)

① *Iron* ← *hemosiderin* *نقطة بنية وبيضاء*
② —

Lipofuscin pigment (Lipochrome)

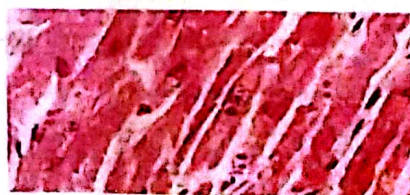
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تعريف **Definition:** Yellowish brown Intracellular, Lipid-derived pigment, due to cell aging (waer and tear)

Pathogenesis: lipid peroxidation + waste products → accumulated in lysosomes

Brown atrophy of the heart :

- **Cause:** Senility (aging) - Starvation - Chronic ischemia
- **Microscopic:** cell size: reduced , cytoplasm: yellow brown pigment around nucleus,



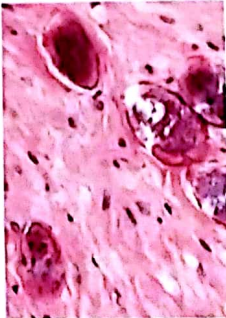
Pathological Calcification

Pathological deposition of calcium salts in tissues other than bone and teeth.

Gross:

- Color : white
- Consistency: Hard granular, or chalky

Micro: Basophilic in color, intracellular or extracellular

	Dystrophic calcification	Metastatic calcification
Tissue	Dead tissue, Degenerated tissue	Normal tissue
Serum Ca	Normal (9-11)	Elevated > 11
Causes and sites	Dead: • Necrosis (.....) • Necrotic cancer • Dead fetus • Dead parasites and ova • Blood thrombus • Blood hematoma Degenerated: PASS • Psammoma bodies • Atherosclerosis & Monckeberg • Scar • Stroma of tumors  	Elevation of serum calcium : <u>1-Increased absorption:</u> • ↑ Vitamin D (e.g. sarcoidosis) macrophage activate v. D precursor • ↑ Ca (milk alkali syndrome) <u>2-Increased bone resorption:</u> • ↑ Parathyroid 1ry - 2ry to R.F. - parathyroid like proteins • Bone tumors (multiple myeloma) • Immobilization Serum Ca → deposits in alkaline tissues • Renal tubular membrane • Gastric mucosa • Alveolar wall • Blood vessels rarely synovium
Mechanism	• Increased intracellular <u>Ca</u> • Increased intracellular <u>phosphate</u> <u>Calcium phosphate</u> stones	Increased serum calcium and phosphate

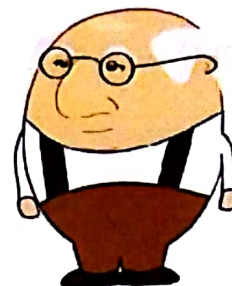


Psammoma bodies : Rounded laminated bodies , seen in some tumors (Menengioma – Papillary carcinomas)

Cell aging

Factors affecting aging

- Internal (Telomere length)
- Environmental (Inhaled pollution)
- Diet (obesity , alcohol)
- Diseases (Atherosclerosis, hypertension, DM)



Mechanism

- Shortening of telomere in gene by telomerase after fixed number of cell divisions → decreased senescence (arrest of cell cycle)
- Accumulation of free radicals

Quiz

- 1- Enumerate types of metaplasia
- 2- Define Agenesis
- 3- Discuss mechanism of apoptosis
- 4- Compare Necrosis vs Apoptosis
- 5- Enumerate causes of fatty liver
- 6- Compare Dry vs Moist gangrene
- 7- Compare Dystrophic vs metastatic calcification
- 8- Discuss mechanism of cell aging
- 9- Complete:
 - a. Consistency of fat necrosis.....
 - b. Coagulative necrosis is caused by.....
 - c. Type of necrosis in brain infarction.....
 - d. Hyperplasia is defined as.....
 - e. Precancerous adaptation includes.....
 - f. Cystitis glandularis is an example of
 - g. Barret's eosophagitis is an example of.....
 - h. Intestinal gangrene isgangrene
 - i. Line of demarcation is defined as.....
 - j. Russel body is an example of.....
- 10- True/False
 - a. Cellular swelling is irreversible
 - b. Cell membrane is damaged in apoptosis
 - c. Diabetic gangrene is dry
 - d. Calcified atheroma is an example of metastatic calcification